IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

ATTY.'S DOCKET: EISENBACH4A

In re Application of:	)	Art Unit: 1643
	)	
EISENBACH et al.	)	Examiner: L. A. Bristol
	)	
Appln. No.: 10/524,787	)	Washington, D.C.
	)	
Date Filed: September 23, 2005	)	Confirmation No. 8693
	)	
For: TUMOR ASSOCIATED ANTIGEN…	)	

DECLARATION UNDER 37 CFR §1.132

Honorable Commissioner for Patents
U.S. Patent and Trademark Office
Customer Service Window
Randolph Building, Mail Stop
401 Dulany Street
Alexandria, VA 22314

Sir:

I, Lea EISENBACH, hereby declare and state as follows:

I am a co-inventor of the above-identified application and my educational and professional experience is presented in the curriculum vitae attached hereto.

I understand that there are three 35 U.S.C. §112, first paragraph, lack of enablement rejections that are maintained in the above-identified application and I will address below all three enablement issues together, as they are very closely related.

The examiner asserts that only peptides were loaded onto a single kind of APC, RMA/HHD/B7.1, and no examples of a gene-loaded APC are shown. The examiner further states that applicants' arguments rely on common knowledge for asserting art-recognized genetic engineering techniques to express peptides from APCs without any indicia of authority for this statement.

Attached hereto as an indicia of authority is the review by Lesterhuis et al., "Dendritic cell vaccines in melanoma: From promise to proof?", *Critical Reviews in Oncology/Hematology* 66:118-134 (2008). In section 7, pages 122-124, dendritic cell (DC) antigen-loading is reviewed and discussed. Although this review article was published in 2008, many of the articles cited and referred to therein were published prior to the 2003 filing date of the present application. For instance, on page 124, left column of Lesterhuis' review article, Sullenberger et al., "Emerging clinical applications of RNA", *Nature* 418:252-258 (2002), as reference [100], is cited for a novel antigen-loading technique using transfection of DC with RNA, and Nair et al., "Induction of tumor-specific cytotoxic T lymphocytes in cancer patients by autologous tumor RNA-transfected dendritic cells", *Ann. Surg.* 235:540-549 (2002), as reference [101], is cited for using tumor-derived RNA for antigen-loading. Two

additional references [106] and [107], Van Tendeloo et al., "Highly efficient gene delivery by mRNA electroporation in human hematopoietic cells: superiority to lipofection and passive pulsing of mRNA and to electroporation of plasmid cDNA for tumor antigen loading of dendritic cells", *Blood* 98:49-56 (2001) and Ponsaerts et al., "mRNA-electroporated mature dendritic cells retain transgene expression, phenotypical properties and stimulatory capacity after cryopreservation", *Leukemia* 16:13224-1330 (2002), demonstrate that RNA electroporation is highly efficient for DC transfection as a means for antigen-loading. As reported in Nair et al., "Induction of carcinoembryonic antigen (CEA)-specific cytotoxic T-lymphocyte responses *in vitro* using autologous dendritic cells loaded with CEA peptide or CEA RNA in patients with metastatic malignancies expressing CEA", *Int. J. Cancer* 82:121-124 (1999), as reference [112], equal immunogenicity was demonstrated for peptide- and RNA-pulsed DC (for antigen-loading) in colorectal cancer patients.

As positive results were obtained with the peptide-pulsed APC antigen-loading method used in Example 1 of the present specification and in the Tirosh et al. (2007) paper, previously made of record with the declaration filed September 18, 2008, one of ordinary skill in the art would conclude that the well known methods of loading APCs are interchangeable and

would fully expect the same results using any other method of antigen-loading of APCs.

The Eisenbach reference listed and attached with my previous declaration filed September 18, 2008, was presented to show that minor variants of the positively selected peptides in the present specification may occur without substantially affecting the ability to be presented by APCs and to activate CTLs.

The examiner refers to applicants wishing to obtain patent coverage for any peptide of 8-10 amino acids in length from any known or yet to be discovered TAA protein expressed by any human colon carcinoma cells formulated into any antigen presenting cell composition. However, it should be pointed out that the TAA protein must be overexpressed by human colon carcinoma cells, and the genes that are overexpressed by human colon carcinoma cells are known and available in the literature. They were known at the time the present invention was made. This is how applicants first found them. Note, for example, in the present specification on page 17, paragraph [0040] and in the first page, right column, second paragraph of the Tirosh et al. (2007) paper, referring to use of the data of Zhang et al., "Gene Expression profiles in normal and cancer cells", *Science* 276:1268-1272 (1997), a copy of which is attached hereto.

The putative TAA peptides derived from these known overexpressed genes were obtained by structural analysis, *in silico*, to find the peptides that are most likely to fit into an APC pocket. The model used was described in the present specification and in the Tirosh et al. (2007) paper. The experimental procedure of narrowing the 500 putative peptides to the ones that actually have the properties required by the claim are all described in the specification and in the Tirosh et al. (2007) paper and could be reproduced by any one of ordinary skill in the art with a lot of routine and tedious work but no undue experimentation. Nothing inventive would need to be done in reproducing the experiments. One of ordinary skill in the art reading the specification would certainly expect that if the experiment were to be repeated, one would end up with the same peptides. The experiments in Example 1 of the present specification and in the Tirosh et al. (2007) paper were conducted only with respect to HLA-A2 haplotype antigen-presenting cells and a mouse model having the human HLA-A2 gene. As the HLA-A2 haplotype is less than half of the population, based on the guidance provide by the present specification, one of ordinary skill in the art is enabled for not only the peptides specifically found in the experiments of Example 1 of the specification, which are expected to be the best peptides for the HLA-A2 population,

but also for whatever peptides would best be applicable to other APC haplotypes. This person of ordinary skill in the art would simply start from the 26 overexpressed genes and model them against the APC haplotype in question to find the putative peptides that are most likely to fit into the pocket of that APC and then repeat the tests using mice that are transgenic for that particular human haplotype of interest and using the APCs of that haplotype. This same person of ordinary skill in the art would then expect that other TAA peptides would be found and these other TAA peptides should also be part of the present invention as they can be found without undue experimentation.

The examiner states that the peptide examples are not further enabling for the "infinite genus of peptide classes encompassed by the claims." However, this statement is unfair. The starting materials are known and the 26 overexpressed proteins in human colon carcinoma cells are known. The models for finding the potentially APC-binding TAA peptides are described in the specification. The number of TAA peptides that have to be screened for any given HLA haplotype are finite and relatively small. Thus, the examiner's characterization is unjustified.

The examiner holds that the specification and the Tirosh et al. (2007) reference do not teach the amount of

experimentation required for obtaining the TAA peptides from human 1-8D interferon inducible protein 2. As shown in Table 2 in the present specification and in the Tirosh et al.(2007) paper, 24 HLA-A2.1 peptides from human 1-8D interferon inducible protein 2 were screened to identify the three TAA peptides. It should not be necessary to explain how much work was involved as the work is all described in the specification and one of ordinary skill in the art can see from the guidance provided what needs to be done. Clearly, it is a lot of work to screen the approximately 500 peptides from the 26 colorectal-associated genes indicated in Table 2 of the present specification and in Table 1 of Tirosh et al. (2007), but still, 500 is a relatively small finite number that can be screened without undue experimentation.

Nevertheless, from the guidance provided in the present specification, it is clear that the first step of finding overexpressed genes in human colon carcinoma has already been done and does not have to be repeated. The second step of finding the putative TAA peptides would have to be repeated with a different model. Models are available for some different haplotypes and this modeling is done by computer program and does not take a substantial amount of effort - a couple of weeks at most. Any university laboratory for PhD level studies in biotechnology would be equipped to do

this, and therefore this would not involve undue experimentation. The third step is for the 500, or however many there are, putative TAA peptides to be synthesized for screening. These peptides can be ordered from companies that do contract peptide synthesis, and while the cost may be expensive, obtaining such peptides is most certainly routine. The next step is to check for binding of the putative TAA peptides to APCs, which is done *in vitro* using APCs isolated from mice or by producing the APCs to the desired haplotype recombinantly. The binding tests must be done at different dilutions in 96-well plates. In general, 500 TAA peptides can be screened in 4 or 5 weeks using the standard equipment available in such a laboratory. Of course, for this routine screening, the more people working on the project, the shorter the time needed for screening.

At the same time or following the screening of the putative TAA peptides, CTLs that can be used in the following step are produced by immunizing a mouse, humanized with the HLA haplotype being tested, with human carcinoma extract and isolating the CTLs from the spleen. Then each of the peptides that were found positive in the previous screen are loaded on the APC of the desired haplotype and incubated together with the CTLs to see if the APCs die. Although this represents a lot of tedious work and although it also may take six months

or more for a single PhD student to complete the testing, one of ordinary skill in the art would clearly recognize that it is something that a PhD student could be taught to do and that it could be done without undue experimentation. Again, the more people working on the project, the shorter the time involved. No inventive steps are necessary to complete such a screen. This screening narrows the 500 putative TAA peptides down to probably 20-30 different TAA peptides. The next step is to take the TAA peptides that were positive in the previous step, load them onto APCs and actually immunize the mice with the peptide-loaded APCs. One would again isolate the CTLs and test for specificity against human colon carcinoma cells. The amount of time depends on how many people are working on it and how many animals are being used simultaneously. It took my laboratory one year to do this but it could have been done faster with more people and more funding. The work was certainly not "undue," but rather involved standard routine experimental work. In the experiments reported in Example 1 of the present specification, we ended up with seven TAA peptides that were positive, all seven of which fall within the scope of the present claims. For the further experiments, my laboratory happened to decide to concentrate on three of the identified TAA peptides.

In short, while there may be a substantial amount of work involved in repeating the process described above (with guidance provided in the present specification) on another haplotype, it would not take undue experimentation to do so and to find TAA peptides which would be usable in that portion of the population.

The undersigned declares further that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

6/16/2009 /Lea Eisenbach/
Date Lea EISENBACH

G:\BN\Y\YEDA\eisenbach4a\pto\2009-06-16Declaration132.doc

LEA EISENBACH, PhD

CURICULUM VITAE

EDUCATION

1962-66 Technical High School of Engineering, Tel Aviv University, Tel Aviv, Israel. Subject of thesis: Immunospecific antibodies to nucleosides.

1966 Awarded degree in Practical Chemical Engineering.

1966-69 Undergraduate studies in the Department of Chemistry, Tel Aviv University.

1969 Awarded B.Sc. degree, Tel Aviv University.

1969-71 Graduate studies towards M.Sc. degree, Department of Biochemistry, Tel Aviv University. Subject of thesis: "The Modification of Porcine Carboxypeptidase B by P-azo-benzenearsonate." Supervisor: Prof. M. Sokolovsky.

1972 Awarded M.Sc. degree, Tel Aviv University.

1973-78 Graduate studies towards Ph.D. degree at the Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel. Subject of thesis: "On the Mechanism of Glucose-6-Phosphate Dehydrogenase Regulation in Mouse Liver." Supervisor: Prof. G. Yagil.

1979 Awarded Ph.D. degree, Feinberg Graduate School of the Weizmann Institute of Science.

1978-79 Postdoctoral training, The Institute for Enzyme Research, Madison, Wisconsin, USA. Research associate with Prof. M. Nomura. Research interests: Effects and mechanisms of ppGpp on transcription of E. coli ribosomal RNA operons in an in vitro system.

1979-80 Postdoctoral training, Department of Biochemistry, University of Wisconsin, Madison, Wisconsin, USA Position: Research Associate with Profs. D.L. Nelson and C. Kung. Research interests: The biochemistry of behavior of Paramecium tetraurelia. Mapping of proteins in the ciliary membrane which are functional in regulation of swimming.

POSITIONS

1980-83 Research Associate with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel. Research interest: Tumor metastases.

1983-86 Staff Assistant Scientist with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.

1986-1989 Staff Associate Scientist, with Professor Michael Feldman, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.

1989-1994 Staff Senior Scientist, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.

1994-1995 Associate Professor, Dept. of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel.

1995 Associate Professor, Dept. of Immunology, The Weizmann Institute of Science, Rehovot, Israel (rearrangement of staff in departments).

MISCELLANEOUS

1966-67 Army service.

1970-72 Non-degree studies in the Department of Education, Tel Aviv University.

1972 Teaching certificate in Chemistry.

1969-70 Teaching, chemistry, physics, high school level.

1969-72 Teaching, mathematics at "Seminar Hakibbutzim", Tel Aviv (College level).

1969-71 Teaching assistant, Department of Biochemistry, Tel Aviv University.

1971-73 Research assistant in a joint program for the Department of Chemical Immunology of the Weizmann Institute and the Immunology Laboratory at the Beilinson Hospital, Petah-Tikva, Israel.

1983 Mini-sabbatical, Dr. Robert Gallo, NCI, Bethesda, Maryland. Subject: Oncogene Expression in Metastases.

1988 Mini-sabbatical, Dr. Eli Gilboa, Memorial Sloan Kettering, New York, Subject: Gene transfer by retroviral vectors.

1995-1998 Secretary general of the Israeli Immunological Society.

1997 Review Board Free University, Brussels, Belgium.

1998 Review committee ACSBI, YY, TCRF-UICC.

1998-2001 President of the Israel Immunological Society.

PROFESSIONAL SOCIETY MEMBERSHIPS

Israel Immunological Society.
Secretary general (1995-1998, President 1998 -2001).

International Metastasis Research Society.

American Association for Cancer Research.

European society for gene therapy.
Chairperson of the committee of gene therapy and immunotherapy of cancer (2000-2003).

UICC.
(Reviewer Board 98-03).

EDITORIAL BOARDS

Gene Therapy.
Karol Sikora, Bob Williamson, Joseph Glorioso (Eds). Macmillan Magazines Ltd.

Clinical and experimental metastasis.
Suzanne A Eccles, Garth L Nicolson, Tatsuro Irimura (Eds), Kluwer Pub.

REVIEWER (Journals)

PNAS
Journal of Immunology
Oncogene
International Journal of Cancer
Clinical and Experimental Metastasis
British Journal of Cancer
International Immunology
Gene Therapy
Cancer gene therapy
Human gene therapy
European J Immunology
European J Cancer

Immunology letters
FEBS journal
Cancer letters
Molecular Medicine

INVITED PRESENTATIONS (2003-2008)

Keystone Symposia, Tumor Immunology (C5) February, 17-23, 2003, Keystone, USA.

The 34th symposium of princess Takamatsu cancer research fund, November 11-15,2003, Tokyo, Japan.

Vaccines, present and future. November 27-29, 2003, Dead Sea resort, Israel

CapCure Israel Scientific Retreat, January 13-16, 2004, Ein Gedi, Israel.

The 33ed annual meeting of the Israel Immunological Society, February 25-26, 2004, Bar-Ilan University, Israel.

The Seventh International Conference of Anticancer Research, October 25-30, 2004, Corfu, Greece.

Israel Vaccine Research Initiative (IVRI), June 21, 2005, Jerusalem, Israel.

Immune Mediated diseases: from Theory to therapy. October 3-8, 2005, Moscow, Russia.

Cancer, Metastasis, AIDS and Immunotherapy, ISF workshop in memory of Professor Michael Feldman. March 13-14, 2006. Rehovot Israel. (Meeting chairperson).

Tumor Immunology, May 17-21, 2006. Halle, Germany.

The scientific legacy of Shraga Segal, a symposium to his memory, February 21, 2007, Beer-Sheba, Israel.

The 14^{th} international conference of cryosurgery, the 1^{st} international conference on cryoimmunology, November 3-6, 2007. Beijing, China.

Cancer-a cellular and molecular view, M.D. Moross Cancer Institute of the WIS and the McGill Cancer Centre, McGill University, Montreal,
October 26-27, 2008. Rehovot, Israel.

GRANTS AND AWARDS (2002-2008)

ICA (1/2001-12/2002)

MHC class I and class II restricted antigens in immunotherapy of cancer.

Ornest Family Foundation (3/2001-2/2002)
Carcinoma TAAs-biology and immunotherapy.

Sterenberg Fund (8/2001-7/2002)
1-8D gene and colon carcinoma.

ISF(10/2001-9/2005)
The role of interferon inducible gene 1-8D in tumorigenicity and immunogenicity of colon cancer.

Minerva (1/2002-12/2004)
The role of interferon-inducible 1-8D gene in tumorigenicity and immunogenicity of colon cancer.

Levin Fund (2/2002-12/2002)
Glycopeptides as targets for immunotherapy of breast and ovarian carcinomas.

Lombroso Grant for Clinical Cancer Research (4/2002-3/2003)
Novel colon carcinoma TAAs-biology and immunotherapy.

Moross Institute for Cancer Research (4/2002-3/2003)
Novel colon carcinoma TAAs-biology and immunotherapy

Ministry of industry and commerce (Nofar) (8/2002-7/2003)
Anti colon carcinomas vaccines based on novel human tumor associated antigen 1-8D peptides.

Ornest Family Foundation (9/2002-8/2003)
Novel anti prostate cancer vaccines based on tumor associated antigen peptides.

ICRF (9/2002-8/2004)
The role of 1-8 family of interferon inducible gene in carcinogenesis and immunotherapy.

ICA (1/2003-12/2004)
Peptides and glycopeptides as targets for anti cancer immunotherapeutic approaches in breast and ovarian carcinomas.

Weizmann Institute-Excellence Center (10/2003-9/2004)
Characterization of new human caricinoma antigens (TAAs) and of respective T cell memory and effector cell responses.

Horowitz (1/2004-2/2005)
Novel anti prostate cancer vaccines based on tumor associated antigen peptides.

Lewis Family Charitable Trust (3/2004-2/2008)
Anti colon-carcinoma vaccines based on tumor associated antigens (TAA) peptides.

Horowitz (1/2005-12/2005)
The role of normal and mutant 1-8D interferon inducible gene in carcinogenesis and immunotherapy.

ICRF (9/2005-8/2007)
The role of the 1-8 interferon inducible genes in tumor progression.

ISF (10/2005-9/2009)
Small interferon stimulated genes: Role in cancer and immunity.

Ministry of health (3/2007-2/2009)
The effect of SNPs in tumor-associated antigens on the immunogenicity of peptide based vaccines.

Moross Institute for Cancer Research (7/2007-6/2008)
T-cell receptor evolution for tumor immunotherapy.

Yeda research and development (7/2007-6/2008)
Preparation of glioma immunotherapy clinical trial-glioma separation and characterization, and immune monitoring of patients.

Lewis Family Charitable Trust (3/2008-2/2009)
Anti colon-carcinoma vaccines based on tumor associated antigens (TAA) peptides.

STUDENTS

Ph.D. degree

Gelber Cohava (graduated 1989)
Kushtai Gil (graduated 1989)
Do Myoung-Sool (graduated 1993)
Fitzer-Attas Cheryl (graduated 1993)
Porgador Angel (graduated 1993)
Katz Anne (graduated 1994)
Plaksin Daniel (graduated 1994)
Mandelboim Ofer (graduated 1996)
Simons Arnon (in collaboration with Prof. V. Rotter, graduated 1998)
Feigelson Sara (in collaboration with Prof. Z. Eshhar, graduated 1999)
El-Shami Khaled (graduated 2000)
Carmon Lior (graduated 2001)
Tirosh Boaz (graduated 2002)
Bar-Haim Erez (graduated 2003)
Lee Sung-Hyung (graduated 2005)
Machlankin Arthur (graduated 2005)
Farago Marganit (left 2003)
Volovitz Ilan (in collaboration with Prof. I. Cohen, graduated 2007)
Goldberger Ofir (graduated 2008)
Daniel Vered (graduated 2008)
Avraham Efrat
Cohen Noam (in collaboration with Dr Steffen Jung)
Boxbaum Yonathan
Cafri Gal

Post-docs

Stepensky David (2003-2005)
Avrahami Dorit (2004-present)
Daniel-Carmi Vered (2008-present)

M.Sc. degree

Plaksin Daniel (graduated 1987)
Gubbay John (graduated 1988)
Porgador Angel (graduated 1989)
Mandelboim Ofer (graduated 1991)
Yamit-Hezi Ayala (graduated 1991)
Erel Naomit (graduated 1993)
Feigelson Sara (graduated 1993)
Kopcow Hernan (graduated 1996)
Morag Tamar (in collaboration with Prof. M. Fridkin, graduated 1996)
Bar-Haim Erez (graduated 1997)
Popovich Dan (graduated 1998)

Shachar Hadas (graduated 1998)
Stabinsky Michal (graduated 1998)
Volovitz Ilan (graduated 2000)
Priel Irene (graduated 2001)
Goldberger ofir (graduated 2002)
Avraham Efrat (graduated 2003)
Daniel Vered (graduated 2003)
Finkel Eran (graduated 2003)
Windrech Ruty (graduated 2004)
Guttman Chen (graduated 2005)
Sheiket Helena (graduated 2005)
Tavori Hagai (graduated 2007)
Karp Noga (graduated 2008)
Reuven Eliran
Azulay Meir
Bordigary Giovanna
Vidiborsky Zoya

LEA EISENBACH, PhD

PUBLICATIONS 72-08

1. Sokolovsky, M., Eisenbach, L. (1972). Porcine carboxypeptidase B Arsanilizocarboxypeptidase, spectral and functional consequences of modification of tyrosine-248. Eur J Biochem 25:483-490.

2. Eisenbach, L., Yagil, G. (1975). Purification of glucose-6-phosphate dehydrogenase from induced and repressed mouse liver. Isr J Med Sci 11:1175.

3. Eisenbach, L., Shimron, F., Yagil, G. (1976). The effect of age on the regulation of glucose-6-phosphate dehydrogenase in mouse liver. Exp Gerontol. 11:63-71

4. Eisenbach, L., Eisenbach, M. (1979). Electrophoretic mobility of membrane fragments on a sucrose gradient. Application to isolated purple membrane fragments from Halobacterium halobium. Anal Biochem 92:228-232.

5. Eisenbach, L., De Baetselier, P., Katzav, S., Segal, S., Feldman, M. (1983). Immunogenic control of metastatic competence of cloned tumor cell populations. Symp Fundam. Cancer Res. 36:101-121.

6. Eisenbach, L., Ramanathan, R., Nelson, DL. (1983). Biochemical studies of the excitable membrane of Paramecium tetraurelia. IX. Antibodies against ciliary membrane proteins. J Cell Biol 97:1412-1420.

7. Eisenbach, L., Segal, S., Feldman, M. (1983). MHC imbalance and metastatic spread in Lewis Lung carcinoma clones. Int J Cancer 32:113-120.

8. Eisenbach, L., Hollander, N., Greenfeld, L., Yakor, H., Segal, S., Feldman, M. (1984). The differential expression of H-2K versus H-2D antigens, distinguishing low metastatic from high metastatic clones, is correlated with the immunogenic properties of the tumor cells. Int J Cancer 34:567-5731.

9. Eisenbach, L., De Baetselier, P., Katzav, S., Segal, S., Feldman, M. (1984). Immunogenetic control of metastatic competence of cloned tumor cell populations. In: "Cancer Invasion and metastasis: Biologic and Therapeutic Aspects," eds Nicolson GL, Milas L, Raven Press, New York, pp 101-121.

10. Eisenbach, L., Segal, S., Feldman, M. (1985). Proteolytic enzymes in tumor metastasis. I. Plasminogen activator in clones of Lewis lung carcinoma and T10 sarcoma. J. Natl. Cancer Inst. 74:77-86.

11. Eisenbach, L., Segal, S., Feldman, M. (1985). Proteolytic enzymes in tumor metastasis. II. Collagenase type IV activity in subcellular fractions of cloned tumor populations. J. Natl. Cancer Inst. 74:87-93.

12. Eisenbach, L., Hollander, N., Segal, S., Feldman, M. (1985). The differential expression of class I MHC antigens controls the metastatic properties of tumor cells. Transpl. Proc. 17:729-734.

13. Eisenbach, L., Feldman, M. (1985). Genes and antigens controlling tumor metastasis. In: Haematology and Blood Transfusion, Modern Trends in Leukemia, VI (Neth R, Gallo R, Greaves M, Jenkins A, eds). Vol 29, Springer Verlag, Berlin, pp. 449-507.

14. Eisenbach, L., Feldman, M. (1986). Enzymes, receptors, oncogenes in high- and low-metastatic tumor clones. In: New Experimental Modalities in the Control of Neoplasia (Chandra P, ed.) Plenum Press, New York, pp 57-70.

15. Eisenbach, L., Katzav, S., Feldman, M. (1986). Immunomodulation of tumor metastasis. Ibidem, pp 81-90.

16. Dvorat, A., Scharf, J., Eisenbach, L., Gershon, D. (1986). G6PD molecules devoid of catalytic activity are present in the nucleus of the rat lens. Exp. Eye Res. 42:489-496.

17. Eisenbach, L., Kushtai, G., Plaksin, D., Feldman, M. (1986). MHC genes and oncogenes control the metastatic phenotype of tumor cells. Cancer Rev. 5:1-8.

18. Barzilay, J., Kushtai, G., Plaksin, D., Feldman, M., Eisenbach, L. (1987). Expression of major histocompatibility class I genes in differentiating leukemic cells is temporally related to activation of c-fos protooncogene. Leukemia 1:198-204.

19. Feldman, M., Eisenbach, L. (1987). Molecular controls of tumor metastasis. In: Accomplishments in Cancer Research 1986 (Fortner JG, Rhoads JE, eds.) JR Lippincott Co., Philadelphia, pp 194-200.

20. Kushtai, G., Barzilay, J., Feldman, M., Eisenbach, L. (1988). The c-fos proto-oncogene in murine 3LL carcinoma clones controls the expression of MHC genes. Oncogene 2:119-128.

21. Plaksin, D., Gelber, C., Feldman, M., Eisenbach, L. (1988). Reversal of the metastatic phenotype in Lewis lung carcinoma cells following transfection with syngeneic H-2K^b gene. Proc. Natl. Acad. Sci. USA 85:4463-4467.

22. Feldman, M., Eisenbach, L. (1988). Genes controlling the metastatic phenotype. Cancer Sur. 7:555-572.

23. Eisenbach, L., Gubbay, J., Gelber, K., Kushtai, G., Feldman, M. (1988). Do oncogenes play a role in tumor metastasis? In: Cancer Metastasis: Biological and Biochemical Mechanisms and Clinical Aspects (Hellman K, Liotta LA, Prodi G, eds.) Plenum Publ. Corp. New York, pp 281-291.

24. Feldman, M., Plaksin, D., Gelber, C., Kushtai, G., Eisenbach, L. (1988). Control of MHC genes that regulate the metastatic phenotype of tumor cells. In: Cancer Metastasis: Biological and Biochemical Mechanisms and Clinical Aspects (Hellman K, Liotta LA, Prodi G, eds). Plenum Publ. Corp., New York, pp 281-291.

25. Feldman, M., Gelber, C., Plaksin, D., Kushtai, G., Eisenbach, L. (1988). The reversal of the metastatic phenotype by gene transfer. Proc. CIBA Found. Symp. on Metastasis, London pp 170-189.

26. Feldman, M., Eisenbach, L. (1988). The metastatic phenotype of cancer cells. Sci. Am. Vol. 256 No. 11:60-85.

27. Feldman, M., Plaksin, D., Gelber, C., Kushtai, G., Eisenbach, L. (1988). Control of MHC genes that regulate the metastatic phenotype of tumor cells. Adv Exp Med Biol 233:109-117.

28. Gelber, C., Plaksin, D.. Vadai, E., Feldman, M., Eisenbach, L. (1989). The abolishment of metastasis formation by tumor cells transfected with "foreign" H-2K genes. Cancer Res. 49:2366-2372.

29. Tzehoval, E., Dagan, S., Eisenbach, L., Atsmon, J., Feldman, M. (1989). Immunogenic properties of macrophage hybridomas. Eur J Immunol 19:89-96.

30. Martinez, RD., Eisenbach, L., Feldman, M. (1989). Cytotoxic and proliferative effect of tobacco products on Lewis lung adenocarcinoma cells and spleen lymphocytes. Allergol Immunopathol 17:257-261.

31. Porgador, A., Feldman, M., Eisenbach, L. (1990). H-2K^{b} transfection of B16 melanoma cells result in reduced tumorigenicity and metastatic competence. J. Immunogenet 16:291-303.

32. Kushtai, G., Feldman, M., Eisenbach, L. (1990). c-fos transfection of 3LL tumor cells turns on MHC gene expression and consequently reduces their metastatic competence. Int. J. Cancer 45:1131-1136.

33. Feldman, M., Eisenbach, L. (1990). Metastases unveiled (in Hebrew). Mada Vol. 34, No. 4, pp. 174-179.

34. Porgador, A., Brenner, B., Vadai, E., Feldman, M., Eisenbach, L. (1991). Immunization by g-IFN treated B16-F10.9 melanoma cells protects against metastatic spread of the parental tumor. Int J Cancer Sup 6: 54-60.

35. Gelber, C., Eisenbach, L., Feldman, M., Goodenow, R .(1991). T cell subset analysis of 3LL tumor rejection. Int J Cancer Sup 6: 69-72.

36. Brosh, N., Lotan, M., Eisenbach, L., Brocke, S. Tartakovsky, B. (1991). Fertility impairment and improved fetal survival induced by a tumor cell line in mice. Am. J. Reprod. Immunol. 26: 47-51.

37. Cordon-Cardo, C., Fuks, Z., Eisenbach, L., Feldman, M. (1991). Expression of HLA-A, B,C antigens on primary and metastatic tumor cell populations of human carcinomas. Cancer Res , 51:6372-6380.

38. Eisenbach, L., Feldman, M. (1991). Tumor cell surface determinants in host immunity against metastases. Semin Cancer Biol. 3:179-188.

39. Feldman, M., Eisenbach, L. (1991). MHC class I genes controlling the metastatic phenotype of tumor cells. Semin Cancer Biol. 5:337-346.

40. Amsterdam, A., Eisenbach, L., Suh, BS., Plehn-Oujovich, D., Keren-Tal, I., Dantes, A. (1991). Possible role for Ha-ras expression in inducible steroidogenesis in immortalized Ganulosa cell lines. In: Proc. NATO Adv Res Workshop on the Superfamily of Ras-Related Genes. Spandidos SA, ed. Plenum Press, pp. 227-236.

41. Eisenbach,, L. (1991). MHC+X, complex formation and antibody induction. Book review. Molec. Immunol. 28, 561-562.

42. Feldman, M., Eisenbach, L. (1991). MHC class I genes controlling the metastatic phenotype of tumor cells. In: Klein E, ed: "The Role of MHC Class I and II Antigen Expression for Immune Surveillance against Tumors", W.B. Saunders, Pub., Vol 2, pp. 337-346.

43. Fridkis-Hareli, M., Abel, L., Eisenbach, L., Globerson, A. (1992). Differentiation patterns of CD4/CD8 thymocyte subsets in cocultures of fetal thymus and lymphohemopoietic cells from c-fos transgenic and normal mice. Cell. Immunol.141: 279-292.

44. Katz, A., Feldman, M., Eisenbach, L. (1992). The use of ^{35}S-methionine as a target cell label in long-term cytotoxic assays. J Immunol Meth 149: 255-260.

45. Orr-Urtreger, A., Bedford, MT., Do, MS., Eisenbach, L., Lonai, P. (1992). Developmental expression of the α receptor platelet-derived growth factor which is deleted in the embryonic lethal Patch mutation. Development 115: 289-303.

46. Mandelboim, O., Feldman, M., Eisenbach, L. (1992). H-2K double transfected tumor cells as anti-metastatic vaccines in heterozygous mice: implications to the T cell repertoire. J. Immunol. 148: 3666-3673.

47. Do, MS., Fitzer-Attas, C., Gubbay, J., Greenfeld, L., Feldman, M., Eisenbach, L. (1992). Mouse PDGFα receptor: sequence, tissue-specific expression and correlation with metastatic phenotype. Oncogene 7:1567-1575.

48. Porgador, A., Tzehoval, E., Katz, A., Vadai, E., Revel, M., Feldman, M., Eisenbach, L. (1992). IL-6 gene transfection into 3LL tumor cells suppresses the metastatic phenotype and confers immunotherapeutic competence against parental metastatic cells. Cancer Res. 52:3679-3686.

49. Suh, BS., Eisenbach, L., Amsterdam, A. (1992). Adenosine 3',5'-monophosphate suppresses metastatic spread in nude mice of steroidogenic rat granulosa cells transformed by Simian virus-40 and Ha-ras oncogene. Endocrinology 131:526-532.

50. Plaksin, D., Gelber, C., Eisenbach, L. (1992). H-2D^b gene transfer into high-metastatic D122 cells results in tumor rejection in allogeneic recipients, but does not affect metastasis in syngeneic recipients: implications to mechanisms of allorejection. Int. J. Cancer. 52:771-777.

51. Gelber, C., Eisenbach, L., Feldman, M., Goodenow, R. (1992). T cell subset analysis of Lewis lung carcinoma tumor rejection: Heterogeneity of effectors and evidence for negative regulatory lymphocytes correlating with metastasis. Cancer Res. 52:6567-6575.

52. Porgador, A., Revel, M., Feldman, M., Eisenbach, L. (1992). Tumor cells transferred with IL-6 gene reversed their metastatic phenotype, and were used as anti-metastatic cellular vaccines. In: Revel M (ED): IL-6, Physiopathology and Clinical Potential. New York: Serono Symposia Publications, Raven Press, pp. 265-272.

53. Revel, M., Chen, L., Novick, D., Shulman, L., Harrochs, S., Chebath, J., Haran-Ghera, N., Eisenbach, L., Katz, A., Feldman, M., Givon, T., Slavin, S. (1992). Antitumor potential of human rIL-6: studies in cell cultures and in murine tumor models in vivo. Ibidem pp. 241-254.

54. Eisenbach, L., Plaksin, D., Porgador, A., Mandelboim, O., Yamit-Hezi, A., Fitzer-Attas, C., Do, MS., Feldman, M. (1992). Genes and antigens controlling tumor metastasis. In: Rabes H, Peters PE, Munk K (eds): Metastasis: Basic Research and its Clinical Applications. Contrib Oncol. Basel, Karger, Vol. 44, pp. 1-12s.

55. Fitzer-Attas, C., Feldman, M., Eisenbach, L. (1993). Expression of functionally intact PDGFa receptors in highly metastatic 3LL Lewis lung carcinoma cells. Int. J. Cancer 53:315-322.

56. Porgador, A., Gansbacher, B., Bannerji, R., Tzehoval, E., Gilboa, E., Feldman, M., Eisenbach, L. (1993). Antimetastatic vaccination of tumor-

bearing mice with IL-2 gene inserted tumor cells. Int J Cancer 53:471-477.

57. Katz, A., Porgador, A., Shulman, L., Revel, M., Feldman, M., Eisenbach L. (1993). Abrogation of B16 melanoma metastasis by prolonged low dose IL-6 therapy. J. Immunotherapy 13:98-109.

58. Fenig, E., Nordenberg,.J., Lurie, H., Porgador, A., Feldman, M., Eisenbach, L. (1993). Amplification of the expression of the major histocompatibility class-I antigens by inducers of differentiation and gamma interferon in murine and human solid tumor cell lines. Int. J. Oncol. 2:279-282.

59. Porgador, A., Bannerji, R., Watanabe, Y., Feldman, M., Gilboa, E., Eisenbach, L. (1993). Anti-metastatic vaccination of tumor-bearing mice with two types of γIFN gene inserted tumor cells. J. Immunol. 150:1458-1470.

60. Katz, A., Shulman, LM., Revel, M., Feldman, M., Eisenbach, L. (1993). Combined therapy with IL-6 and inactivated tumor cells suppresses metastasis in mice bearing 3LL lung carcinomas. Int. J. Cancer 53:812-818.

61. Plaksin, D., Baeurele, P., Eisenbach, L. (1993). KBF1-p50 homodimers are suppressors of MHC Class I expression in 3LL tumor cells. J. Exp. Med. 177:1651-1662.

62. Rabizadeh, E., Shaklai. M., Nudelman. A., Eisenbach. L. (1993) Rapid alteration of c-myc and c-jun expression in leukemic cells induced to differentiate by a butyric acid prodrug. FEBS 328:225-229.

63. Porgador, A., Tzehoval, E., Vadai, E., Feldman, M., Eisenbach, L. (1993). Immunotherapy via gene therapy: comparison of the effects of tumor cells transduced with IL2, IL6 or γIFM genes. J. Immunotherapy 14:191-201.

64. Fridkin, M., Rosen, O., Landsmann, P., Preciado-Patt, L., Tzehoval, E., Eisenbach, L., Hahn, T., Levantomsky, D., Pras, M., Spirer, L. (1993). Enzymatic processing of serum proteins - A route to peptides associated with host defense. In: "Peptide Chemistry" S. Sakakibara Ed. Escom Science Pub. pp. 637-642.

65. Feldman, M., Eisenbach, L. (1993). MHC control of growth and metastatic properties of tumor cells. In: Solheim BG, Ferrone S, Möller E. (eds), "The HLA System in Clinical Transplantation. Basic Concepts and Importance.". Springer Berlin Heidelberg New York. pp. 185-198.

66. Yamit-Hezi, A., Plaksin, D., Eisenbach, L. (1994). c-fos and c-jun overexpression in malignant cells reduces their tumorigenic and

metastatic potential and affects their MHC class I gene expression. Oncogene 9:1065-1079.

67. Porgador, A., Feldman, M., Eisenbach, L. (1994). Immunotherapy via gene therapy. Nat. Immunity 13:113-130.

68. Mandelboim, O., Berke, G., Fridkin, M., Feldman, M., Eisenstein, M., Eisenbach, L. (1994). CTL induction by a tumour-associated antigen octapeptide derived from a murine lung carcinoma. Nature 369:67-71.

69. Fitzer-Attas, C., Eldar, H., Eisenbach, L., Livneh, E. (1994). The expression of PDGF-α but not PDGF-β receptors is suppressed in Swiss/3T3 fibroblasts over-expressing protein kinase C-α. FEBS Lett. 342:165-170.

70. Plaksin, D., Porgador, A., Vadai, E., Feldman, M., Schirrmacher, V., Eisenbach, L. (1994). Effective anti metastatic melanoma vaccination with tumor cells transfected with MHC genes and/or infected with Newcastle Disease virus (NDV). Int. J. Cancer 59:796-801.

71. Mandelboim, O., Vadai, E., Fridkin, M., Katz-Hillel, A., Feldman, M., Berke, G., Eisenbach, L. (1995). Regression of established murine carcinoma metastases following vaccination with tumor associated antigen peptides. Nature Medicine 1:1179-1183.

72. Feldman, M., Porgador, A., Mandelboim, O., Plaksin, D., Eisenbach, L. (1994). Tumor invasion and metastasis: Immunotherapy via gene therapy. In: Lung Cancer Frontiers in Science and Treatment. G. Motta ed. Grafica L.P. Pub. pp. 67-82.

73. Revel, M., Katz, A., Eisenbach, L., Feldman, M., Haran-Ghera, N., Harroch, S., Chebath, J. (1995). Interleukin-6 effects on tumor models in mice and on the cellular regulation of transcription factor IRF-1. Ann NY Acad Sci 762:342-355.

74. Porgador, A., Tzehoval, E., Vadai, E., Feldman, M., Eisenbach, L. (1995). Combined vaccination, by MHC class-I and IL-2 gene transduced melanoma cells, syngergizes the cure of post-surgical established lung metastases. Cancer Res. 55:4941-4949.

75. Mandelboim, O., Vadai, E., Feldman, M., Eisenbach, L. (1995). Expression of two H-2K genes, syngeneic and allogeneic, as a strategy for potentiating immune recognition of tumor cells. Gene Therapy 2:757-765.

76. Eisenbach, L. (1996). Curing metastases gene and peptide therapy. Attempts to understand metastases formation. III. Therapeutic Approaches for Metastasis Treatment. U. Gunthest, P.M. Schlag, W. Birchmeier (eds). Current Topics in Microbiology and Immunology 213:85-100.

77. Rabizadeh, E., Shaklai, M., Eisenbach, L., Nudelman, A., Rephaeli, A. (1996). Esterose inhibitors diminish the modulation of gene expression by butyric acid derivative pivaloyloxymethylbutyrate (AN-9). Isr. J. Med. Sci. 32:1186-1191.

78. Simons, A., Melamed-Bessudo, C., Wolkowicz, R., Sperling, J., Sperling, R., Eisenbach, L., Rotter, V. (1997). PACT: cloning and characterization of a cellular p53 binding protein that interacts with Rb. Oncogene 14:145-155.

79. Fitzer-Attas, CJ., Do, MS., Vadai, E., Feigelson, S., Feldman, M., Eisenbach, L. (1997). Modification of PDGF receptor expression or function alters the metastatic phenotype of 3LL cells. Oncogene 15:1545-1553.

80. Oh, J.W., Katz, A., Harroch, S., Eisenbach, L., Revel, M., Chebath, J. (1997). Unmasking by soluble IL-6 receptor of IL-6 effect on metastatic melanoma: Growth inhibition and differentiation of B16-F10.9 cells. Oncogene 15:569-577.

81. Mandelboim, O., Bar-Haim, E., Vadai, E., Fridkin, M., Eisenbach, L. (1997). Identification of shared tumor associated antigen peptides between two spontaneous lung carcinomas. J. Immunol. 159:6030-6036.

82. Inbal, B., Cohen, O., Polak-Charcon, S., Kopolovic, J., Vadai, E., Eisenbach, L., Kimchi, A. (1997). DAP-kinase is a tumor suppressor gene which links the control of apoptosis to metastasis. Nature 390:180-184.

83. Eisenbach, L., Mandelboim, O., Bar-Haim, E., Carmon, L., Copcow, H., El-Shami, K., Paz, A., Popovic, D., Vadai, E., Tzehoval, E., Feldman, M., Fridkin, M. (1998). Tumor associated antigen peptides as anti-metastatic vaccines. LIPS 5:323-328.

84. Popovic, D., El-Shami, KM., Vadai, E., Feldman, M., Tzehoval, E.,Eisenbach, L. (1998). Antimetastatic vaccination against Lewis lung carcinoma with autologous tumor cells modified to express murine interleukin 12. Clin Exp Metastasis 16:623-632.

85. El-Shami, KM., Tirosh, B., Bar-Haim, E., Carmon, L., Vadai, E., Fridkin, M., Feldman, M., Eisenbach, L. (1999). MHC class I-restricted epitope spreading in context of tumor rejection following vaccination with a single immunodominant CTL epitope. Eur J Immunol 29:3295-3301.

86. Tirosh, B., El-Shami, KM., Vaisman, N., Carmon. L., Bar-Haim, E., Vadai, E., Feldman, M., Fridkin, M., Eisenbach, L. (1999). Immunogenicity of H-2Kb-low affinity, high affinity, and covalently - bound peptides in anti-tumor vaccination. Immunol Lett 70:21-28.

87. El-Shami, KM., Tzehoval, E., Vadai, E., Feldman,M., Eisenbach, L. (1999). Induction of antitumor immunity with modified autologous cells expressing membrane-bound murine cytokines. J Interfeon Cytokine Res. 19:1391-1401.

88. El-Shami, KM., Tirosh, B., Popovic, D.,Carmon, L., Tzehoval, E., Vadai, E., Feldman, M., Eisenbach, L. (2000). Induction of antitumor immunity by proteasome-inhibited syngeneic fibroblasts pulsed with a modified TAA peptide. Int J Cancer 85:236-242.

89. Carmon, L., El-Shami, KM., Paz, A., Pascolo, S., Tzehoval, E., Tirosh, B., Koren, R., Feldman, M., Fridkin, M., Lemonnier, FA., Eisenbach, L. (2000). Novel breast-tumor-associated MUC1-derived peptides: characterization in Db-/-xbeta2microglobulin null mice transgenic for a chimeric HLA-A2.1/Db-beta2microglobulin single chain. Int J Cancer 85:391- 397.

90. Eisenbach, L., El-Shami, KM. (2000). Anti-tumor vaccination: immunotherapy Vs Autoimmunity. In Cancer and Autoimmunity, Shoenfeld, Y. Gershwin, ME. (Eds). Elsevier Science (pub). pp 397-408.

91. Carmon, L., Tzehoval, E., Vadai, E., Feldman, M., Eisenbach, L. (2000). Anti-tumor vaccination in heterozygous congenic F1 mice: presentation of TAA by two parental class I alleles J Immunotherapy, 23:344-352.

92. Eisenbach, L., Bar-Haim, E.,El-Shami, KM. (2000). Antiiumor vaccination using peptide based vaccines. Immunol Lett. 74:27-34.

93. Zier, K., Johnson, K., Maddux, JM., Sung, M., Mandeli, J., Eisenbach, L., Schwartz, M. (2000). Antigen specific secretion of IFN-gammaagainst tumor peptides prepared from fresh tumor tissue in colon cancer patients. J. Immunol . Methods 241:61-68.

94. Tirosh, B., Fridkin, M., Tzehoval, E., Vadai, E., Lemonnier, FA., Eisenbach, L. (2000). Antigenicity and immunogenicity of an intracellular delivery system of MHC class I epitopes, which bypasses the proteasome processing. J immunotherapy, 23:622-630.

95. Zier, K., Maddux, JM., Johnson, K., Sung, M., Mandeli, J., Eisenbach, L., Schwartz, M. (2001). Surrogate markers of antitumor responses: in vitro activation of T cells by autologous tumor peptides. Clin Cancer Res 7:818s-821s.

96. Weiss-Feigelson, S., Fitzer-Attas, C., Eisenbach, L. (2001) The role of platelet derived growth factor (PDGF) and its receptors in cancer and metastasis. in Growth factors and their receptors in cancer metastasis.WG. Jiang, K. Matsumoto, T. Nakamura (Eds), Kluwer Academic Publishers. pp 167-186.

97. Barnea, E., Beer, I., Patoka, R., Ziv, T., Kessler, O., Tzehoval, E., Eisenbach, L., Zavazava, N., Admon, A. (2002). Analysis of endogenous peptides bound by soluble MHC class I molecules: A novel approach for identifying tumor specific antigens. Eur. J. Immunol. 32:213-222.
http://www3.interscience.wiley.com/cgi-bin/abstract/89012506/ABSTRACT

98. Markel, G., Katz, G., Arnon, TI., Lotem, M., Bar-Haim, E., Blumberg, RS., Drize, O., Eisenbach, L., Mandelboim, O. (2002). Expression of CD66a (CEACAM1) on human melanoma and NK cells: A novel class I MHC-independent inhibitory mechanism of human NK cell cytotoxicity. J. immunol. 168:2803-2810.
http://www.jimmunol.org/cgi/content/full/168/6/2803

99. Carmon, L., Bobilev-Priel, I., Brenner, B., Bobilev, D., Paz, A., Bar-Haim, E., Tirosh, B., Klein, T., Fridkin, M., Lemonnier, FA., Tzehoval, E., Eisenbach, L. (2002). Characterization of novel breast tumor-associated BA46-derived peptides in Db-/-xbeta2 microglobulin (beta2m)-/- null mice transgenic for chimeric HLA-A2.1/Db-beta2m single chain. J. Clin. Invest. 110:453-462.
http://www.jci.org/cgi/content/full/110/4/453

100. Cohen, CJ., Hoffman, N., Farago, M., Hoogenboom, HR., Eisenbach, L., Reiter, Y. (2002). direct detection and quantitation of a distinct T cell epitope derived from tumor-specific epithelial cell-associated mucin (MUC1) using human recombinant antibodies endowed with the antigen-specific, major histocompatibility complex-restricted specificity of T cells. Cancer Res. 62: 5835–5844.
http://cancerres.aacrjournals.org/cgi/content/full/62/20/5835

101. Papo, N., Shahar, M., Eisenbach, L., Shai, Y. (2003). A novel lytic peptide composed of D, L amino acids selectively kills cancer cells in culture and in mice. J. Biol. Chem. 278:21018-21023.
http://www.jbc.org/cgi/content/full/278/23/21018

102. Qimron, U., Madar, N., Bar-Haim, E., Eisenbach, L., Staats, HF., Porgador, A.(2002). Anti-cancer mucosal vaccines: therapeutic efficacy against cancers growing in mucosal organs. In: I.P. Witz (ed), Proceedings of the 2nd international conference on tumor microenvironment. Monduzzi Editore, Bologna, Italy. pp. 197-206.

103. Margalit, O., Eisenbach, L., Amariglio, N., Kaminski, N., Harmelin, A., Pfeffer, R., Shohat, M., G Rechavi, G., Berger, R. (2003). Overexpression of a set of genes, including WISP-1, common to pulmonary metastases of both mouse D122 Lewis lung carcinoma and F10.9 melanoma cell lines. Br. J. Cancer. 89:314-319.
http://www.nature.com/bjc/journal/v89/n2/abs/6600977a.html;jsessionid=C4E97D6CC43B63C27CE85CD23497C5D3

104. Denkberg, G, Lev, A, Eisenbach, L, Benhar, I, Reiter, Y. (2003). Selective targeting of melanoma and APCs using a recombinant antibody with TCR-like specificity directed toward a melanoma differentiation antigen. J. Immunol. 171:2197-2207.
http://www.jimmunol.org/cgi/content/full/171/5/2197

105. Lee, SH., Bar-Haim, E., Goldberger, O., Reich-Zeliger, S., Vadai, E., Tzehoval, E. Eisenbach, L. (2004). Expression of FasL by tumor cells does not abrogate anti-tumor CTL function. Immunol. Lett. 91:119-126.
http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T75-4B6C9WS-1&_user=46001&_coverDate=02%2F15%2F2004&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=C000004958&_version=1&_urlVersion=0&_userid=46001&md5=74d73574c1c8c867c6b2436dec914060

106. Markel G, Mussaffi H, Ling KL, Salio M, Gadola S, Steuer G, Blau H, Achdout H, de Miguel M, Gonen-Gross T, Hanna J, Arnon TI, Qimron U, Volovitz I, Eisenbach L, Blumberg RS, Porgador A, Cerundolo V, Mandelboim O. (2004). The mechanisms controlling NK cell autoreactivity in TAP2-deficient patients. Blood 103: 1770-8. Epud 2003 Nov 6.
http://bloodjournal.hematologylibrary.org/cgi/content/full/103/5/1770

107. Lee, SH., Bar-Haim, E., Machlenkin, A., Goldberger, O., Volovitz, I., Vadai, E., Tzehoval, E., Eisenbach, L. (2004). In vivo rejection of tumor cells dependent on CD8 cells that kill independently of perforin and FasL. Cancer Gene Therapy 11:237-248.
http://www.nature.com/cgt/journal/v11/n3/pdf/7700678a.pdf

108. Qimron, U., Paul, L., Bar-Haim, E., Bloushtain, N., Eisenbach, L., Staats, HF., Porgador A. (2004). Non-replicating mucosal and systemic vaccines: Quantitative and qualitative differences in the Ag-specific CD8+ T cell population in different tissues. Vaccine 22:1390-1394.
http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6TD4-4B7YFNS-5&_user=46001&_coverDate=03%2F29%2F2004&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=C000004958&_version=1&_urlVersion=0&_userid=46001&md5=f9986207a4707bb3fe039b013f30d126

109. Bar-Haim, E., Paz, A., Machlenkin, A., Hazzan, D., Tirosh, B., Carmon, L., Brenner, B., Vadai, E., Mor, O., Stein, A., Lemonnier, FA., Fridkin, M., Cytron, S., Tzehoval, E., Eisenbach, L. (2004). MAGE-A8-overexpression in transitional cell carcinoma of the bladder: identification of two tumour-associated antigen peptides. British J. Cancer 91:398-407.
http://www.nature.com/bjc/journal/v91/n2/pdf/6601968a.pdf

110. Berko, D., Carmi, Y., Cafri, G., Ben-Zaken, S., Sheikhet, H., Tzehoval, E., Eisenbach, L., Margalit, A., Gross, G. (2005). Membrane-anchored

beta 2-microglobulin stabilizes a highly receptive state of MHC class I molecules. J. Immunol. 174:2116-2123.
http://www.jimmunol.org/cgi/content/full/174/4/2116

111. Machlenkin, A., Paz, A., Bar-Haim, E., Goldberger, O., Finkel, E., Tirosh, B., Volovitz, I., Vadai, E., Lemonnier, F., Tzehoval, E., Eisenbach, L. (2005). Human CTL Epitopes Prostatic Acid Phosphatase-3 and Six-Transmembrane Epithelial Antigen of Prostate-3 as Candidates for Prostate Cancer Immunotherapy. Cancer Res. 65: 6435-6442.
http://cancerres.aacrjournals.org/cgi/content/full/65/14/6435

112. Machlenkin, A., Goldberger, O., Tirosh, B., Paz, A., Volovitz, I., Bar-Haim, E., Lee, SH., Vadai, E., Tzehoval, E., Eisenbach, L. (2005). Combined dendritic cell cryotherapy of tumor induces systemic antimetastatic immunity. Clin. Cancer Res. 11:4955-4961.
http://clincancerres.aacrjournals.org/cgi/content/full/11/13/4955

113. Stepensky, D., Tzehoval, E., Vadai, E., Eisenbach, L. (2006). O-glycosylated vs. non-glycosylated MUC1-derived peptides as potential targets for cytotoxic immunotherapy of carcinoma. Clinical and Experimental Immunology. 143:139-149.
http://www.blackwell-synergy.com/doi/abs/10.1111/j.1365-2249.2005.02965.x

114. Margalit, A., Sheikhet, HM., Carmi, Y., Berko, D., Tzehoval, E., Eisenbach, L., Gross, G. (2006) Induction of anti-tumor immunity by CTL epitopes genetically linked to membrane-anchored β2 microglobulin. J. Immunol. 176:217-224.
http://www.jimmunol.org/cgi/content/full/176/1/217

115. Machlenkin, A., Rosenfeld, R., Volovitz, I., Vadai, E., Lev,A., Paz, A., Goldberger, O., Reiter, Y., Tzehoval, E., Benhar, I., Eisenbach, L. (2007). Active immunization with PAP-3, a novel human prostate cancer peptide, inhibits carcinoma development in HLA transgenic mice. Cancer Immunoloy immunotherapy. Cancer Immunol. Immunother. 56:217-26. Epub 2006 Jun 1.
http://www.springerlink.com/content/gw478359v6042882/

116. Issaeva, I., Zonis,Y., Rozovskaia,T., Orlovsky, K., Croce, CM., Nakamura, T., Mazo, A., Eisenbach, L., Canaani, E. (2007). Knockdown of ALR (MLL2) reveals ALR target genes and leads to alterations in cell adhesion and growth. Mol. Cell. Biol. 27, 1889–1903.
http://mcb.asm.org/cgi/content/full/27/5/1889?view=long&pmid=17178841

117 Tirosh, B., Daniel Carmi, V., Carmon, L., Paz, A., Lugassy, G., Vadai, E., Machlenkin, A., Bar Haim, E., Do, MS., Ahn, IS., Fridkin, M., Tzehoval, E., Eisenbach, L. (2007). "1-8 interferon inducible gene

family" - putative colon carcinoma associated antigens. Br. J. Cancer 97:1655-1663.

118. Cohen, N., Stolarsky-Bennun, M., Amir-Kroll, H., Margalit, R., Nussbaum, G., Cohen-Sfady, M., Pevsner-Fischer, M., Fridkin, M., Bercovier, H., Eisenbach, L., Jung, S., Cohen, IR. (2008). Pneumococcal capsular polysaccharide is immunogenic when present on the surface of macrophages/dendritic cells: TLR-4 signaling induced by a conjugate vaccine or by LPS is required. J. Immunol. 180:2409-2418.

119. Machlenkin, A., Uzana, R., Frankenburg, S., Eisenbach, L., Pitkovski, J., Gorodetsky, R., Peretz, T., Lotem, L. (2008). Membrane capture by tumor-reactive T cells: A novel concept in identification of functional cytotoxic T lymphocytes. Cancer Res. 68:2006-2013.

120. Goldberger, O., Volovitz, I., Machlenkin, A., Vadai, E., Tzehoval, E., Eisenbach, L. (2008). Exuberated numbers of tumor-specific T cells result in tumor escape. Cancer Res. 68:3450-3457.

121. Zhang QJ, Li XL, Wang D, Huang XC, Mathis JM, Duan WM, Knight D, Shi R, Glass J, Zhang DQ, Eisenbach L, Jefferies WA. (2008). Trogocytosis of MHC-I/peptide complexes derived from tumors and infected cells enhances dendritic cell cross-priming and promotes adaptive T cell responses.
PLoS ONE. 3:e3097.